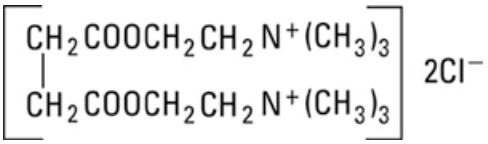
QUELICIN - succinylcholine chloride injection, solution
HOSPIRA, INC.

A short-acting depolarizing skeletal muscle relaxant.
Abboject® Syringe
Fliptop Vial
R_x only

WARNING
RISK OF CARDIAC ARREST FROM HYPERKALEMIC RHABDOMYOLYSIS
There have been rare reports of acute rhabdomyolysis with hyperkalemia followed by ventricular dysrhythmias, cardiac arrest and death after the administration of succinylcholine to apparently healthy pediatric patients who were subsequently found to have undiagnosed skeletal muscle myopathy, most frequently Duchenne's muscular dystrophy.
This syndrome often presents as peaked T-waves and sudden cardiac arrest within minutes after the administration of the drug in healthy appearing pediatric patients (usually, but not exclusively, males, and most frequently 8 years of age or younger). There have also been reports in adolescents.
Therefore, when a healthy appearing infant or child develops cardiac arrest soon after administration of succinylcholine, not felt to be due to inadequate ventilation, oxygenation or anesthetic overdose, immediate treatment for hyperkalemia should be instituted. This should include administration of intravenous calcium, bicarbonate, and glucose with insulin, with hyperventilation. Due to the abrupt onset of this syndrome, routine resuscitative measures are likely to be unsuccessful. However, extraordinary and prolonged resuscitative efforts have resulted in successful resuscitation in some reported cases. In addition, in the presence of signs of malignant hyperthermia, appropriate treatment should be instituted concurrently.
Since there may be no signs or symptoms to alert the practitioner to which patients are at risk, it is recommended that the use of succinylcholine in pediatric patients should be reserved for emergency intubation or instances where immediate securing of the airway is necessary, e.g., laryngospasm, difficult airway, full stomach, or for intramuscular use when a suitable vein is inaccessible (see PRECAUTIONS: Pediatric Use and DOSAGE AND ADMINISTRATION).
This drug should be used only by individuals familiar with its actions, characteristics and hazards.

DESCRIPTION

Quelicin (Succinylcholine Chloride Injection, USP) is a sterile, nonpyrogenic solution to be used as a short-acting, depolarizing, skeletal muscle relaxant. See HOW SUPPLIED for summary of content and characteristics of the solutions. The solutions are for I.M. or I.V. use.
Succinylcholine Chloride, USP is chemically designated $C_{14}H_{30}Cl_2N_2O_4$ and its molecular weight is 361.31.
It has the following structural formula:



Succinylcholine is a diquaternary base consisting of the dichloride salt of the dicholine ester of succinic acid. It is a white, odorless, slightly bitter powder, very soluble in water. The drug is incompatible with alkaline solutions but relatively stable in acid solutions. Solutions of the drug lose potency unless refrigerated.
Solutions intended for multiple dose administration contain 0.18% methylparaben and 0.02% propylparaben as preservatives (List Nos. 6629 and 9085). Solutions intended for single-dose administration contain no preservatives. Unused solution should be discarded. Products not requiring dilution (multiple-dose fliptop vial and Abboject syringe) contain sodium chloride to render isotonic. May contain sodium hydroxide and/or hydrochloric acid for pH adjustment. pH is 3.6 (3.0 to 4.5). See table in HOW SUPPLIED for characteristics.
Sodium Chloride, USP, chemically designated NaCl, is a white crystalline compound freely soluble in water.

CLINICAL PHARMACOLOGY

Succinylcholine is a depolarizing skeletal muscle relaxant. As does acetylcholine, it combines with the cholinergic receptors of the motor end plate to produce depolarization. This depolarization may be observed as fasciculations. Subsequent neuromuscular transmission is inhibited so long as adequate concentration of succinylcholine remains at the receptor site. Onset of flaccid paralysis is rapid (less than one minute after intravenous administration), and with single administration lasts approximately 4 to 6 minutes. Succinylcholine is rapidly hydrolyzed by plasma cholinesterase to succinylmonocholine (which possesses clinically insignificant depolarizing muscle relaxant properties) and then more slowly to succinic acid and choline (see PRECAUTIONS). About 10% of

the drug is excreted unchanged in the urine. Succinylcholine levels were reported to be below the detection limit of 2 µg/mL after 2.5 minutes of an IV bolus dose of 1 or 2 mg/kg in fourteen (14) anesthetized patients. The paralysis following administration of succinylcholine is progressive, with differing sensitivities of different muscles. This initially involves consecutively the levator muscles of the face, muscles of the glottis and finally the intercostals and the diaphragm and all other skeletal muscles.
Succinylcholine has no direct action on the uterus or other smooth muscle structures. Because it is highly ionized and has low fat solubility, it does not readily cross the placenta.
Tachyphylaxis occurs with repeated administration (see PRECAUTIONS).
Depending on the dose and duration of succinylcholine administration, the characteristic depolarizing neuromuscular block (Phase I block) may change to a block with characteristics superficially resembling a non-depolarizing block (Phase II block). This may be associated with prolonged respiratory muscle paralysis or weakness in patients who manifest the transition to Phase II block. When this diagnosis is confirmed by peripheral nerve stimulation, it may sometimes be reversed with anticholinesterase drugs such as neostigmine (see PRECAUTIONS). Anticholinesterase drugs may not always be effective. If given before succinylcholine is metabolized by cholinesterase, anticholinesterase drugs may prolong rather than shorten paralysis.
Succinylcholine has no direct effect on the myocardium. Succinylcholine stimulates both autonomic ganglia and muscarinic receptors which may cause changes in cardiac rhythm, including cardiac arrest. Changes in rhythm, including cardiac arrest, may also result from vagal stimulation, which may occur during surgical procedures, or from hyperkalemia, particularly in pediatric patients (see PRECAUTIONS: Pediatric Use). These effects are enhanced by halogenated anesthetics.
Succinylcholine causes an increase in intraocular pressure immediately after its injection and during the fasciculation phase, and slight increases which may persist after onset of complete paralysis (see WARNINGS).
Succinylcholine may cause slight increases in intracranial pressure immediately after its injection and during the fasciculation phase (see PRECAUTIONS).
As with other neuromuscular blocking agents, the potential for releasing histamine is present following succinylcholine administration. Signs and symptoms of histamine mediated release such as flushing, hypotension and bronchoconstriction are, however, uncommon in normal clinical usage.
Succinylcholine has no effect on consciousness, pain threshold or cerebration. It should be used only with adequate anesthesia (see WARNINGS).

INDICATIONS AND USAGE
Succinylcholine chloride is indicated as an adjunct to general anesthesia, to facilitate tracheal intubation, and to provide skeletal muscle relaxation during surgery or mechanical ventilation.

CONTRAINDICATIONS
Succinylcholine is contraindicated in persons with personal or familial history of malignant hyperthermia, skeletal muscle myopathies and known hypersensitivity to the drug. It is also contraindicated in patients after the acute phase of injury following major burns, multiple trauma, extensive denervation of skeletal muscle, or upper motor neuron injury, because succinylcholine administered to such individuals may result in severe hyperkalemia which may result in cardiac arrest (see WARNINGS). The risk of hyperkalemia in these patients increases over time and usually peaks at 7 to 10 days after the injury. The risk is dependent on the extent and location of the injury. The precise time of onset and the duration of the risk period are not known.

WARNINGS
SUCCINYLCHOLINE SHOULD BE USED ONLY BY THOSE SKILLED IN THE MANAGEMENT OF ARTIFICIAL RESPIRATION AND ONLY WHEN FACILITIES ARE INSTANTLY AVAILABLE FOR TRACHEAL INTUBATION AND FOR PROVIDING ADEQUATE VENTILATION OF THE PATIENT, INCLUDING THE ADMINISTRATION OF OXYGEN UNDER POSITIVE PRESSURE AND THE ELIMINATION OF CARBON DIOXIDE. THE CLINICIAN MUST BE PREPARED TO ASSIST OR CONTROL RESPIRATION.
TO AVOID DISTRESS TO THE PATIENT, SUCCINYLCHOLINE SHOULD NOT BE ADMINISTERED BEFORE UNCONSCIOUSNESS HAS BEEN INDUCED. IN EMERGENCY SITUATIONS, HOWEVER, IT MAY BE NECESSARY TO ADMINISTER SUCCINYLCHOLINE BEFORE UNCONSCIOUSNESS IS INDUCED.
SUCCINYLCHOLINE IS METABOLIZED BY PLASMA CHOLINESTERASE AND SHOULD BE USED WITH CAUTION, IF AT ALL, IN PATIENTS KNOWN TO BE OR SUSPECTED OF BEING HOMOZYGOUS FOR THE ATYPICAL PLASMA CHOLINESTERASE GENE.
Hyperkalemia: (SEE BOX WARNING) Succinylcholine should be administered with **GREAT CAUTION** to patients suffering from electrolyte abnormalities and those who may have massive digitalis toxicity, because in these circumstances succinylcholine may induce serious cardiac arrhythmias or cardiac arrest due to hyperkalemia.
GREAT CAUTION should be observed if succinylcholine is administered to patients during the acute phase of injury following major burns, multiple trauma, extensive denervation of skeletal muscle, or upper motor neuron injury (see CONTRAINDICATIONS). The risk of hyperkalemia in these patients increases over time and usually peaks at 7 to 10 days after the injury. The risk is dependent on the extent and location of the injury. The precise time of onset and the duration of the risk period are undetermined. Patients with chronic abdominal infection, subarachnoid hemorrhage, or conditions causing degeneration of central and peripheral nervous systems should receive succinylcholine with **GREAT CAUTION** because of the potential for developing severe hyperkalemia.

Malignant Hyperthermia: Succinylcholine administration has been associated with acute onset of malignant hyperthermia, a potentially fatal hypermetabolic state of skeletal muscle. The risk of developing malignant hyperthermia following succinylcholine administration increases with the concomitant administration of volatile anesthetics. Malignant hyperthermia frequently presents as intractable spasm of the jaw muscles (masseter spasm) which may progress to generalized rigidity, increased oxygen demand, tachycardia, tachypnea and profound hyperpyrexia. Successful outcome depends on recognition of early signs, such as jaw muscle spasm, acidosis, or generalized rigidity to initial administration of succinylcholine for tracheal intubation, or failure of tachycardia to respond to deepening anesthesia. Skin mottling, rising temperature and coagulopathies may occur later in the course of the hypermetabolic process. Recognition of the syndrome is a signal for discontinuance of anesthesia, attention to increased oxygen consumption, correction of acidosis, support of circulation, assurance of adequate urinary output and institution of measures to control rising temperature. Intravenous dantrolene sodium is recommended as an adjunct to supportive measures in the management of this problem. Consult literature references and the dantrolene prescribing information for additional information about the management of malignant hyperthermic crisis. Continuous monitoring of temperature and expired CO_2 is recommended as an aid to early recognition of malignant hyperthermia.

Other: In both adults and pediatric patients the incidence of bradycardia, which may progress to asystole, is higher following a second dose of succinylcholine. The incidence and severity of bradycardia is higher in pediatric patients than adults. Whereas bradycardia is common in pediatric patients after an initial dose of 1.5 mg/kg, bradycardia is seen in adults only after repeated exposure. Pretreatment with anticholinergic agents (e.g., atropine) may reduce the occurrence of bradyarrhythmias.

Succinylcholine causes an increase in intraocular pressure. It should not be used in instances in which an increase in intraocular pressure is undesirable (e.g., narrow angle glaucoma, penetrating eye injury) unless the potential benefit of its use outweighs the potential risk.

Succinylcholine is acidic (pH = 3.5) and should not be mixed with alkaline solutions having a pH greater than 8.5 (e.g., barbiturate solutions).

PRECAUTIONS
(SEE BOX WARNING*)*

General:

When succinylcholine is given over a prolonged period of time, the characteristic depolarization block of the myoneural junction (Phase I block) may change to a block with characteristics superficially resembling a non-depolarizing block (Phase II block). Prolonged respiratory muscle paralysis or weakness may be observed in patients manifesting this transition to Phase II block. The transition from Phase I to Phase II block has been reported in 7 of 7 patients studied under halothane anesthesia after an accumulated dose of 2 to 4 mg/kg succinylcholine (administered in repeated, divided doses). The onset of Phase II block coincided with the onset of tachyphylaxis and prolongation of spontaneous recovery. In another study, using balanced anesthesia (N_2O/O_2/narcotic-thiopental) and succinylcholine infusion, the transition was less abrupt, with great individual variability in the dose of succinylcholine required to produce Phase II block. Of 32 patients studied, 24 developed Phase II block. Tachyphylaxis was not associated with the transition to Phase II block, and 50% of the patients who developed Phase II block experienced prolonged recovery.

When Phase II block is suspected in cases of prolonged neuromuscular blockade, positive diagnosis should be made by peripheral nerve stimulation, prior to administration of any anticholinesterase drug. Reversal of Phase II block is a medical decision which must be made upon the basis of the individual, clinical pharmacology and the experience and judgment of the physician. The presence of Phase II block is indicated by fade of responses to successive stimuli (preferably "train of four"). The use of an anticholinesterase drug to reverse Phase II block should be accompanied by appropriate doses of an anticholinergic drug to prevent disturbances of cardiac rhythm. After adequate reversal of Phase II block with an anticholinesterase agent, the patient should be continually observed for at least 1 hour for signs of return of muscle relaxation. Reversal should not be attempted unless: (1) a peripheral nerve stimulator is used to determine the presence of Phase II block (since anticholinesterase agents will potentiate succinylcholine-induced Phase I block), and (2) spontaneous recovery of muscle twitch has been observed for at least 20 minutes and has reached a plateau with further recovery proceeding slowly; this delay is to ensure complete hydrolysis of succinylcholine by plasma cholinesterase prior to administration of the anticholinesterase agent. Should the type of block be misdiagnosed, depolarization of the type initially induced by succinylcholine (i.e., Phase I block) will be prolonged by an anticholinesterase agent.

Succinylcholine should be employed with caution in patients with fractures or muscle spasm because the initial muscle fasciculations may cause additional trauma.

Succinylcholine may cause a transient increase in intracranial pressure; however, adequate anesthetic induction prior to administration of succinylcholine will minimize this effect.

Succinylcholine may increase intragastric pressure, which could result in regurgitation and possible aspiration of stomach contents.

Neuromuscular blockade may be prolonged in patients with hypokalemia or hypocalcemia.

Reduced Plasma Cholinesterase Activity: Succinylcholine should be used carefully in patients with reduced plasma cholinesterase (pseudocholinesterase) activity. The likelihood of prolonged neuromuscular block following administration of succinylcholine must be considered in such patients (see DOSAGE and ADMINISTRATION).

Plasma cholinesterase activity may be diminished in the presence of genetic abnormalities of plasma cholinesterase (e.g., patients heterozygous or homozygous for atypical plasma cholinesterase gene), pregnancy, severe liver or kidney disease, malignant tumors, infections, burns, anemia, decompensated heart disease, peptic ulcer, or myxedema. Plasma cholinesterase activity may also be

diminished by chronic administration of oral contraceptives, glucocorticoids, or certain monoamine oxidase inhibitors and by irreversible inhibitors of plasma cholinesterase (e.g., organophosphate insecticides, echothiophate, and certain antineoplastic drugs). Patients homozygous for atypical plasma cholinesterase gene (1 in 2500 patients) are extremely sensitive to the neuromuscular blocking effect of succinylcholine. In these patients, a 5 to 10 mg test dose of succinylcholine may be administered to evaluate sensitivity to succinylcholine, or neuromuscular blockade may be produced by the cautious administration of a 1 mg/mL solution of succinylcholine by slow intravenous infusion. Apnea or prolonged muscle paralysis should be treated with controlled respiration.

Drug Interactions:
Drugs which may enhance the neuromuscular blocking action of succinylcholine include: promazine, oxytocin, aprotinin, certain non-penicillin antibiotics, quinidine, β-adrenergic blockers, procainamide, lidocaine, trimethaphan, lithium carbonate, magnesium salts, quinine, chloroquine, diethylether, isoflurane, desflurane, metoclopramide and terbutaline. The neuromuscular blocking effect of succinylcholine may be enhanced by drugs that reduce plasma cholinesterase activity (e.g., chronically administered oral contraceptives, glucocorticoids, or certain monoamine oxidase inhibitors) or by drugs that irreversibly inhibit plasma cholinesterase (see PRECAUTIONS).
If other neuromuscular blocking agents are to be used during the same procedure, the possibility of a synergistic or antagonistic effect should be considered.

Carcinogenesis, Mutagenesis, Impairment of Fertility:
There have been no long-term studies performed in animals to evaluate carcinogenic potential.

Pregnancy:
Teratogenic Effects: Pregnancy Category C.
Animal reproduction studies have not been conducted with succinylcholine chloride. It is also not known whether succinylcholine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Succinylcholine should be given to a pregnant woman only if clearlyneeded.
Nonteratogenic Effects: Plasma cholinesterase levels are decreased by approximately 24% during pregnancy and for several days postpartum. Therefore, a higher proportion of patients may be expected to show increased sensitivity (prolonged apnea) to succinylcholine when pregnant than when nonpregnant.

Labor and Delivery:
Succinylcholine is commonly used to provide muscle relaxation during delivery by caesarean section. While small amounts of succinylcholine are known to cross the placental barrier, under normal conditions the quantity of drug that enters fetal circulation after a single dose of 1 mg/kg to the mother should not endanger the fetus. However, since the amount of drug that crosses the placental barrier is dependent on the concentration gradient between the maternal and fetal circulations, residual neuromuscular blockade (apnea and flaccidity) may occur in the newborn after repeated high doses to, or in the presence of atypical plasma cholinesterase in, the mother.

Nursing Mothers:
It is not known whether succinylcholine is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised following succinylcholine administration to a nursing woman.

Pediatric Use:
Safety and effectiveness of succinylcholine chloride have been established in pediatric patient age groups, neonate to adolescent. There are rare reports of ventricular dysrhythmias and cardiac arrest secondary to acute rhabdomyolysis with hyperkalemia in apparently healthy pediatric patients who receive succinylcholine (see BOX WARNING). Many of these pediatric patients were subsequently found to have a skeletal muscle myopathy such as Duchenne's muscular dystrophy whose clinical signs were not obvious. The syndrome often presents as sudden cardiac arrest within minutes after the administration of succinylcholine. These pediatric patients are usually, but not exclusively, males, and most frequently 8 years of age or younger. There have also been reports in adolescents. There may be no signs or symptoms to alert the practitioner to which patients are at risk. A careful history and physical may identify developmental delays suggestive of a myopathy. A preoperative creatine kinase could identify some but not all patients at risk. Due to the abrupt onset of this syndrome, routine resuscitative measures are likely to be unsuccessful. Careful monitoring of the electrocardiogram may alert the practitioner to peaked T-waves (an early sign). Administration of intravenous calcium, bicarbonate, and glucose with insulin, with hyperventilation have resulted in successful resuscitation in some of the reported cases. Extraordinary and prolonged resuscitative efforts have been effective in some cases. In addition, in the presence of signs of malignant hyperthermia, appropriate treatment should be initiated concurrently (see WARNINGS). Since it is difficult to identify which patients are at risk, it is recommended that the use of succinylcholine in pediatric patients should be reserved for emergency intubation or instances where immediate securing of the airway is necessary, e.g., laryngospasm, difficult airway, full stomach, or for intramuscular use when a suitable vein is inaccessible.

As in adults, the incidence of bradycardia in pediatric patients is higher following the second dose of succinylcholine. The incidence and severity of bradycardia is higher in pediatric patients than adults. Pre-treatment with anticholinergic agents, e.g., atropine, may reduce the occurrence of bradyarrhythmias.

Geriatric Use:
Clinical studies of Quelicin did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently from younger subjects. Other reported clinical experience has not identified differences in responses between the elderly and younger patients.
In general, dose selection for an elderly patient should be cautious, usually starting at the low end of the dosing range, reflecting the greater frequency of decreased hepatic, renal, or cardiac function, and of concomitant disease or other drug therapy.

ADVERSE REACTIONS
Adverse reactions to succinylcholine consist primarily of an extension of its pharmacological actions. Succinylcholine causes profound muscle relaxation resulting in respiratory depression to the point of apnea; this effect may be prolonged. Hypersensitivity reactions, including anaphylaxis, may occur in rare instances. The following additional adverse reactions have been reported: cardiac arrest, malignant hyperthermia, arrhythmias, bradycardia, tachycardia, hypertension, hypotension, hyperkalemia, prolonged respiratory depression or apnea, increased intraocular pressure, muscle fasciculation, jaw rigidity, postoperative muscle pain, rhabdomyolysis with possible myoglobinuric acute renal failure, excessive salivation, and rash.

OVERDOSAGE
Overdosage with succinylcholine may result in neuromuscular block beyond the time needed for surgery and anesthesia. This may be manifested by skeletal muscle weakness, decreased respiratory reserve, low tidal volume, or apnea. The primary treatment is maintenance of a patent airway and respiratory support until recovery of normal respiration is assured. Depending on the dose and duration of succinylcholine administration, the characteristic depolarizing neuromuscular block (Phase I) may change to a block with characteristics superficially resembling a non-depolarizing block (Phase II) (see PRECAUTIONS).

DOSAGE AND ADMINISTRATION
The dosage of succinylcholine should be individualized and should always be determined by the clinician after careful assessment of the patient (see WARNINGS).
Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration whenever solution and container permit. Solutions which are not clear and colorless should not be used.
Adults:
For Short Surgical Procedures: The average dose required to produce neuromuscular blockade and to facilitate tracheal intubation is 0.6 mg/kg Quelicin (succinylcholine chloride) Injection given intravenously. The optimum dose will vary among individuals and may be from 0.3 to 1.1 mg/kg for adults. Following administration of doses in this range, neuromuscular blockade develops in about 1 minute; maximum blockade may persist for about 2 minutes, after which recovery takes place within 4 to 6 minutes. However, very large doses may result in more prolonged blockade. A 5 to 10 mg test dose may be used to determine the sensitivity of the patient and the individual recovery time (see PRECAUTIONS).
For Long Surgical Procedures: The dose of succinylcholine administered by infusion depends upon the duration of the surgical procedure and the need for muscle relaxation. The average rate for an adult ranges between 2.5 and 4.3 mg per minute.
Solutions containing from 1 to 2 mg per mL succinylcholine have commonly been used for continuous infusion. The more dilute solution (1 mg per mL) is probably preferable from the standpoint of ease of control of the rate of administration of the drug and, hence, of relaxation. This intravenous solution containing 1 mg per mL may be administered at a rate of 0.5 mg (0.5 mL) to 10 mg (10 mL) per minute to obtain the required amount of relaxation. The amount required per minute will depend upon the individual response as well as the degree of relaxation required. Avoid overburdening the circulation with a large volume of fluid. It is recommended that neuromuscular function be carefully monitored with a peripheral nerve stimulator when using succinylcholine by infusion in order to avoid overdose, detect development of Phase II block, follow its rate of recovery, and assess the effects of reversing agents (see PRECAUTIONS).
Intermittent intravenous injections of succinylcholine may also be used to provide muscle relaxation for long procedures. An intravenous injection of 0.3 to 1.1 mg/kg may be given initially, followed, at appropriate intervals, by further injections of 0.04 to 0.07 mg/kg to maintain the degree of relaxation required.
Pediatrics: For emergency tracheal intubation or in instances where immediate securing of the airway is necessary, the intravenous dose of succinylcholine is 2 mg/kg for infants and small pediatric patients; for older pediatric patients and adolescents the dose is 1 mg/kg (see BOX WARNING and PRECAUTIONS: Pediatric Use). It is currently known that the effective dose of succinylcholine in pediatric patients may be higher than that predicted by body weight dosing alone. For example, the usual adult IV dose of 0.6 mg/kg is comparable to a dose of 2-3 mg/kg in neonates and infants to 6 months and 1-2 mg/kg in infants up to 2 years of age. This is thought to be due to the relatively large volume of distribution in the pediatric patient versus the adult patient.
Rarely, I.V. bolus administration of succinylcholine in infants and pediatric patients may result in malignant ventricular arrythmias and cardiac arrest secondary to acute rhabdomyolysis with hyperkalemia. In such situations, an underlying myopathy should be suspected.

Intravenous bolus administration of succinylcholine in infants or pediatric patients may result in profound bradycardia or, rarely, asystole. As in adults, the incidence of bradycardia in pediatric patients is higher following a second dose of succinylcholine. Whereas bradycardia is common in pediatric patients after an initial dose of 1.5 mg/kg, bradycardia is seen in adults only after repeated exposure. The occurrence of bradyarrhythmias may be reduced by pretreatment with atropine (see PRECAUTIONS: Pediatric Use).
Intramuscular Use: If necessary, succinylcholine may be given intramuscularly to infants, older pediatric patients or adults when a suitable vein is inaccessible. A dose of up to 3 to 4 mg/kg may be given, but not more than 150 mg total dose should be administered by this route. The onset of effect of succinylcholine given intramuscularly is usually observed in about 2 to 3 minutes.
Compatibility and Admixtures: Succinylcholine is acidic (pH 3.5) and should not be mixed with alkaline solutions having a pH greater than 8.5 (e.g., barbiturate solutions). Admixtures containing 1 to 2 mg/mL may be prepared by adding 1 g Quelicin to 1000 or 500 mL sterile solution, such as 5% Dextrose Injection, USP or 0.9% Sodium Chloride Injection, USP. Admixtures of Quelicin must be used within 24 hours after preparation. Aseptic techniques should be used to prepare the diluted product. Admixtures of Quelicin should be prepared for single patient use only. The unused portion of diluted Quelicin should be discarded.
To prevent needle-stick injuries, needles should not be recapped, purposely bent, or broken by hand.

HOW SUPPLIED
Quelicin® (Succinylcholine Chloride Injection, USP) is supplied as a clear, colorless solution in the following concentrations and packages:

List No.	Container	Size (mL)	mg/mL	mg (total)	mOsmol/mL (calc.)
Single-dose					
8065	Abboject Unit of Use Syringe	5	20	100	0.328
6970	Fliptop Vial	10 in 20	100	1000	0.830
Multiple-dose					
6629	Fliptop Vial	10	20	200	0.338

Refrigeration of the undiluted agent will assure full potency until expiration date. All units carry a date of expiration.
Store in refrigerator 2° to 8°C (36° to 46°F). The multi-dose vials are stable for up to 14 days at room temperature without significant loss of potency.

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